
Tissue-specific expression of Cre recombinase from the Tgfb3 locus.

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Public Summary:

Tgfb3, a member of the TGF-beta superfamily, is tightly regulated, both spatially and temporally, during embryogenesis. Previous mouse knockout studies have demonstrated that Tgfb3 is absolutely required for normal palatal fusion and pulmonary development. We have generated a novel tool to ablate genes in Tgfb3-expressing cells by targeting the promoterless Cre-pgk-Neo cassette into exon 1 of the mouse Tgfb3 gene, which generates a functionally null Tgfb3 allele. Using the Rosa26 reporter assay, we demonstrate that Cre-induced recombination was already induced at embryonal day 10 (E10) in the ventricular myocardium, limb buds, and otic vesicles. At E14, robust recombination was detected in the prefusion palatal epithelium. Deletion of the TGF-beta type I receptor Alk5 (Tgfb1) specifically in Tgfb3 expressing cells using the Tgfb3-Cre driver line lead to a cleft palate phenotype similar to that seen in conventional Tgfb3 null mutants. In addition, Alk5/ Tgfb3-Cre mice displayed hydrocephalus, and severe intracranial bleeding due to germinal matrix hemorrhage.

Scientific Abstract:

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